African Crop Science Journal, Vol. 14. No. 3, pp. 221-230, 2006
Printed in Uganda. All rights reserved

ISSN 1021-9730/2006 $ 4.00

COMBINING ABILITY ANALYSIS OF RESISTANCE TO MOSAIC VIRUS DISEASE IN CASSAVA

Y. LOKKO*, A.G.O. DIXON, S.K. OFFEI[1] and E.Y. DANQUAH[1]
International Institute of Tropical Agriculture (IITA) Ibadan, Nigeria
c/o Lambourn UK Ltd., Carolyn House, 26 Dingwall Road, Croydon CR9 3EE, UK
[1]Department of Crop Science, University of Ghana, P. O. Box 25, Legon-Accra, Ghana
*Current address: Plant Breeding & Geneticist Section, International Atomic Energy Agency, Wagramer Strasse 5, P.O. Box 200, A1400 Vienna, Austria

(Received 1 August, 2005; accepted 24 May, 2006)

ABSTRACT

A North Carolina design II experiment, with three improved cassava (*Manihot esculenta* Crantz) accessions as the female parents, 15 cassava landraces and three improved cassava accessions as the male parents (3 x 18), was evaluated in three environments in Nigeria to determine the mode of gene action and the combining ability, and also to estimate heterosis for resistance to cassava mosaic disease (CMD), in various sources of resistance. General combining ability (GCA) effect due to females and males was significant in each environment; while the GCA effect due to males and the specific combining ability (SCA) effect were significant across environments. The relative magnitude of the total GCA components to the total GCA plus SCA component, however, suggested that GCA was more important than SCA in controlling CMD resistance among the crosses. The test for heterosis was significant in the individual environments; one cross, involving the best general combiner, exhibited significant heterosis for resistance in all three environments. The implication of the findings in breeding for resistance to CMD is discussed.

Key Words: Additive gene, mid-parent heterosis, *Manihot esculenta*, Nigeria

RÉSUMÉ

Une expérience de la Caroline du Nord à conception II avec trois accessions améliorées de manioc *(Manihot esculenta* Crantz) comme parents femelles, 15 races locales et trois accessions améliorées de manioc comme parents males (3X18) a été évaluée au sein de trois environnements au Nigeria en vue de déterminer le mode d'action de gène et la capacité de combinaison et aussi d'estimer mais également en vue d'estimer l'hétérosis pour la résistance à la maladie de la mosa¿que de manioc (CMD) dans différents sources de résistance. L'effet de capacité générale de combinaison (GCA) dû aux males et femelles était significatif dans chaque environnement ; pendant que l'effet GCA dû aux males et l'effet de capacité spécifique de combinaison (SCA) étaient significatifs à travers les environnements. L'ampleur relative de composante de GCA totale au GCA plus composante SCA a cependant suggérée que GCA était plus important que SCA dans la lutte contre CMD au sein des croisés. Le test pour hétérosis était significatif dans les environnements individuels ; un croisé, impliquant le meilleur combinant général, a exposé une hétérosis significative pour la résistance dans tous les trois environnements. Les implications des résultats pour une culture en vue de la résistance au CMD sont examinées.

Mots Clés: Gène additif, hétérosis mi-parent, *Manihot esculenta*, Nigéria

INTRODUCTION

Enhancing resistance to the cassava mosaic virus disease (CMD) is an important breeding objective of cassava, to reduce production losses in Africa, estimated at $2 billion per *annum.* (IITA, 1997). In tackling the early disease challenges in Africa, resistance to CMD was obtained from a cross between cassava and its relative *Manihot glaziovii* Muller von Argau (Nichols, 1947). The resistant genetic stock, accession 58308, was developed from this process and has been the main source of resistance in breeding for resistance to the disease (Hahn *et al.*, 1989). Some of the cassava genotypes derived from 58308 with resistance to CMD and widely cultivated in Africa, are TMS30001, TMS30572, TMS4(2)1425, TMS60142 and TMS90257

Despite the progress made in resistance breeding, there is still need to increase the levels of resistance within the genepool. Epidemics in East Africa, allegedly caused by a recombinant strain of two of the viruses causing the disease (Zhou *et al.*, 1997) resulted in over 2.2 billion tonnes of storage root losses estimated at US $440 million (Thresh *et al.*, 1997). To ensure that durable resistance is maintained, additional sources of resistance with a wider genetic base are sought to diversify resistance that would prove difficult for the pathogen to circumvent.

Some African landraces, the Tropical *Manihot esculenta* series (TME), have been identified as resistant to CMD (Raji, 1995). Landraces which form part of the crops genetic resource and are believed to be a result of spontaneous recombination of cassava with itself and its relatives provide a wealth of new genes for some traits (Gulick *et al.*, 1983; Hershey, 1987). These landraces resistant to CMD could, therefore, serve as new sources of resistance to the disease. However, selection of parents to be incorporated in a breeding programme cannot be based on their performance alone. To exploit different types of gene action involved in the inheritance of a trait, information on their relative magnitude and estimates of combining ability is essential. This would allow the breeder to determine the appropriate breeding strategy to adopt.

In two separate studies, resistance to CMD derived from accession 58308 was reportedly polygenic, recessive and inherited largely in an additive manner (Hahn and Howland, 1972; Jennings, 1977). Neither of these studies, however, considered the effect of environment or seasons in obtaining information on the stability of the genetic effects. Climatic factors that affect vector population, plant growth, and vigour invariably influence the incidence and severity of the disease (Fargette *et al.*, 1994). Therefore, besides incorporating new genes for resistance, the material should be evaluated over several years and in different environments where the virus inoculum from diseased plants is present and whitefly population is high.

An additive and polygenic mode of inheritance has also been reported for some of the African landraces, which represent the new sources of resistance (Lokko *et al.*, 1998). Interestingly, a major dominant gene responsible for resistance in an African landrace was reported (Akano *et al.*, 2002). The objectives of this study were to determine the mode of gene action, and the combining ability for resistance to CMD in the various sources of resistance, and also to estimate heterosis.

MATERIALS AND METHODS

Experimental design and procedure. Six improved cassava accessions and 15 African landraces with varying levels of resistance and susceptibility to CMD (Table 1) had previously been crossed by hand-pollination in IITA research field in Ubiaja, Edo State, Nigeria, in a 3 x 18 North Carolina design II (NCD II). In 1997, the seedlings from the crosses were established at Mokwa, Niger State, Nigeria, to produce woody cuttings for the experiments. The experiments were then established at Mokwa during the 1998 growing season, and in Ibadan, Oyo State, Nigeria, during the 1998 and 1999 growing season. Mokwa is in the southern guinea savannah, while Ibadan is in the forest-savannah transition zone.

The design in each environment was a randomised complete block with two replicates. Plants were established using cuttings approximately 20 cm long and planted in rows (ridges 30 cm high and 10 m long) 1 m apart giving a plant population of 20,000 plants ha^{-1}. Due to differential seed set of the parents, the

number of progenies varied for the different crosses and ranged from 52 to 934 individual genotypes in a cross. To ensure the survival of each F_1 genotype, two cuttings each of a genotype were planted in a replicate. The second cutting was removed at 6 weeks after planting (WAP) when the plants were established. Twenty cuttings of each parental or check genotype were planted in each replicate with the same spacing as the crosses. The experiments were evaluated under rain-fed conditions; no fertiliser or herbicide was applied; and hand-weeding was done when necessary.

Individual plants in each F_1 cross were assessed for their reaction to CMD due to natural infection by whiteflies at 6, 12, and 20 WAP. Assessment was based on the standard 5 point scoring scale for CMD, where a score of 1 indicates no obvious symptom and 5 indicates severe mosaic symptoms and stunting of the entire plant (IITA, 1990).

Preliminary analysis of the data showed that the CMD severity was highest at 12 WAP and the variance of the mean was also highest in this period in all three environments. High CMD incidence at 12 WAP has previously been documented in south-western and southeastern Nigeria (Leuschner 1978; Ogbe *et al.* 1996) and Fargette *et al.* (1994) demonstrated that plants are generally more susceptible to secondary infection by whiteflies during the first 8-12 weeks. Subsequently, genetic analyses of the F_1 crosses were therefore based on CMD severity at 12 WAP, which give an overall impression of the symptom severity potential of a genotype.

Data analysis. Weighted mean disease scores defined as the summation of the product of the frequency count and the value of the disease severity class, divided by the total number of plants evaluated in each replicate in an environment, were estimated for the crosses and

TABLE 1. Description of cassava accessions used as parents and checks, mean CMD score across environments and CMD resistance status

Accession	Pedigree information, local name and origin	Parent	Mean CMD	CMD Resistance Status
TMS30001	Pedigree information lost	Female 1	1.2	R
TMS30555	58308 x Oyarugba dudu	Female 2	2.23	MS
TMS30572+	58308 x Branca de Santa Caterina (OP) +	Female 3	1.55	MR
TMS60142	KR685 OP	Male 1	1.63	MR
TMS90257	58308 x Oyarugba dudu	Male 2	1.48	R
TMS4(2)1425	58308 x Oyarugba fufun	Male 3	1.96	MR
TME1+	Antiota (Ondo, Ondo State, Nigeria)	Male 4	1.48	R
TME2	Odungbo (Opeji, Ogun State, Nigeria)	Male 5	2.68	S
TME4	Atu (Iwo, Kwara State, Nigeria)	Male 6	1.18	R
TME5	Bagiwawa (New Busa, Niger State, Nigeria)	Male 7	1.2	R
TME6	Lapai-1 (Lapai, Niger State, Nigeria)	Male 8	1.37	R
TME7	Oko-Iyawo (New Lapai, Niger State, Nigeria)	Male 9	1.12	R
TME8	Amala (Ireuekpen, Edo State, Nigeria)	Male 10	1.36	R
TME9	Olekanga (Ogbomosho, Oyo State, Nigeria)	Male 11	1.15	R
TME10	Orente (Ogbomosho, Oyo State, Nigeria)	Male 12	2.76	S
TME11	Igueeba (Warri, Delta State, Nigeria)-	Male 13	1.44	R
TME12	Tokunbo (Ibadan, Oyo State, Nigeria)	Male 14	1.39	R
TME14	Abbey Ife (Abbey-Ife, Osun State, Nigeria)	Male 15	1.37	R
TME31	Bakince-Iri (Bahago, Sokoto State, Nigeria)	Male 16	2.77	S
TME41	Danbusa (Kanji, Niger State, Nigeria)	Male 17	3.15	S
TME117+	Isunikankiyan (Ibadan, Oyo State, Nigeria)	Male 18	3.46	S
91/02324+	TME1 OP		1.22	R
$LSD_{5\%}$			0.245	

+Accession used as check; R = resistant; S = susceptible

parental and check genotypes, and used for the statistical analyses. The analyses were done for inter- and intra- environments; with the environments considered random whereas genotypic effects were fixed.

Using the GLM procedure in SAS (SAS, 1999), genotypes were partitioned into the variation due to the test genotypes (parents and crosses) and checks. The test genotypes were then partitioned into the variation due to parents and crosses, and a one-degree of freedom orthogonal contrast (parent versus cross) was performed to test the significance of average mid-parent heterosis. The parents were further partitioned into female, male, resistant, and susceptible parents, and one degree of freedom orthogonal contrasts, female versus male parents, and resistant versus susceptible parents, were used to test for significant variation among the different sets of parents. Similarly, the crosses were partitioned into the variation due to the general combining ability (GCA) effects of the females and males, and the specific combining ability (SCA) effect of their interaction (female x male).

In the individual environment analyses, the genotypic components were tested with the pooled error. For the analysis across environments, the variation due to the environment and the genotype by environment interaction (G x E) effects was also partitioned into its components. Main effects were tested with their respective G x E interaction and the G x E effect was tested with the pooled error.

Ratios of the mean square component associated with the fixed GCA effects of males θ_m and females θ_f to the sum of the mean square components of θ_m, θ_f and the SCA effect were computed to estimate the relative importance of GCA and SCA in predicting progeny performance. The mean square components of these fixed effects were estimated from the variance components of the random effects associated with these fixed effects using the MIVQUE0 option of the VARCOMP procedure in SAS.

Combining ability estimates were based on the methods described by Beil and Atkins (1967) using least square means generated from the GLM analysis. The standard errors of the effects and the test of significance were performed according to Singh and Chuadhary (1985) for a single environment and Cox and Frey (1984) for the combined environment. Pearson correlation was performed on the parent means and GCA effects to compare per se performance of the parents with their GCA effects. Average and individual mid-parent and high-parent heterosis were estimated and tested for significance, as described by Dixon *et al.* (1990) using least square means.

Mean CMD severity score of the parental and check accessions were also derived form the least square means and used to classify the parents and checks (Table 1). Parental and check means were converted to a whole integer, and accessions with a score of '1' were classified as resistant, '2' were moderately resistant, '2' and '3' were classified as moderately susceptible, and susceptible accessions had scores from '3' to '5'.

RESULTS

The variation due to environments, replicates nested in environments and genotypes were highly significant (Table 2). All components of G x E were significant, except for the check by environment, the parent contrast, female versus male by environment (F vs M x E), and the SCA by environment (SCA x E) interactions.

The variation among the test genotypes was due to significant variation ($P<0.01$) among the parents and crosses in Ibadan 1998 and 1999; and across environments. In the Mokwa 1998 environments, significant variation among the test genotypes was due to the parents only. In the individual environments significant variation among the parents was due to the four parent types, female, male, resistant and susceptible; while across environments, variation among the parents was due to the male parent only. The parent contrast female versus male (F vs M) was significant ($P<0.05$) in Mokwa 1998 and across environments, while the contrast resistant versus susceptible parents, was significant ($P<0.01$) in all three environments and across environments.

The GCA effect of the males and SCA effect of the females by male interaction were significant in Ibadan in 1999, and across environments. The GCA effect of the females was also significant in Ibadan 1998 and 1999. The parent versus cross contrast, which is a test of average heterosis, was significant in the individual environments but its

effect across environments was not significant. In the environment with significant variation among the crosses, the ratio of the mean square component associated with GCA to the sum of mean square components associated with to GCA and SCA, was 0.76 across environments, 0.84 in Ibadan 1998 and 0.96 in Ibadan 1999. In the Ibadan 1999 environment and across environments, GCA and parents means were also significant ($P<0.05$) and positively correlated.

In this study, a negative GCA effect was desirable for resistance. Negative GCA effects

TABLE 2. Analysis of variance for CMD severity at 12 weeks after planting (WAP) among all genotypes in 3 x 18 NCD II mating scheme, evaluated in three environments

Source of variation			Across environments		df (†)	Individual environments		
						Mokwa 1998	Ibadan 1998	Ibadan 1999
			df	MS		MS	MS	MS
Environment (E)			2	59.17**				
Replicates within E			3	1.40**	1	1.18**	2.98**	0.05**
Genotypes in population (G)			78	1.39**	78(77)	0.36**	0.49**	1.06**
Checks (Ck)			3	7.72**	3(2)	2.46**	2.71**	3.48**
Test Genotypes (TG)			74	1.12**	74	0.30**	0.38**	0.96**
Ck versus TG			1	1.32	1	2.57**	1.97**	1.56**
	Parent (P)		20	3.17**	20	0.91**	0.89**	1.90**
		Female (F)	2	1.64	2	0.55**	0.38*	1.24**
		Male (M)	17	3.51**	17	0.99**	0.99**	2.08**
		F versus M	1	0.46*	1	0.23*	0.07	0.19
		Susceptible (S)	5	1.08	5	0.36**	0.68**	0.51**
		Resistant (R)	14	0.29	14	0.11**	0.16*	0.63**
		R versus S	1	53.98**	1	15.15**	12.55**	26.47**
	Crosses (C)		53	0.31**	53	0.04	0.19**	0.37**
		F (GCA)	2	1.40	2	0.18**	2.28**	0.42**
		M (GCA)	17	0.57*	17	0.06*	0.14	0.91**
		F x M (SCA)	34	0.11*	34	0.02	0.09	0.13**
	P vs C		1	3.10	1	1.77**	0.54*	13.22**
GXE			155	0.28**				
Ck x E			5	0.07				
TG x E			148	0.26**				
Ck vs TG x E			2	2.71**				
	P x E		40	0.26**				
		F x E	4	0.26**				
		M x E	34	0.28**				
		F vs M x E	2	0.14				
		S x E	10	0.36**				
		R x E	28	0.36**				
		R vs S x E	2	1.01**				
	C x E		106	0.15**				
		F (GCA) x E	4	0.54*				
		M (GCA) x E	34	0.27*				
		F x M (SCA) x E	68	0.07				
	P vs CxE		2	6.20**				
Error (genotypes)			233	0.07	78(77)	0.07	0.09	0.04
Error (crosses)			159	0.06	53	0.08	0.08	0.03
$\frac{GCA}{GCA + SCA}$				0.76		0.84	0.94	1.00

(†) df of genotypes and checks in Mokwa 1998 where check, 91/02324 was missing
* Significantly different from zero at the 0.5 probability level
** Significantly different from zero at the 0.01 probability level

were estimated for the improved accession TMS30572 in all environments, but its effect was significant only in the Ibadan 1998 environment (Table 3). The improved accessions TMS4(2)1425, TMS60142 and TMS90257 also had negative GCA effect in all three environments, but this was significant for TMS4(2)1425 and TMS60142 in Ibadan 1999, and for TMS90257 in Ibadan 1999 and across environments. Negative and significant GCA effects were also detected for the resistant landraces TME8 and TME9 in the Ibadan 1999 environment.

The susceptible landrace TME2 had a significant and positive GCA in all and across environments, while the susceptible landraces TME31, TME41, and TME117 had significant and positive GCA effects only in the Ibadan 1999 environment. The resistant accession TMS30001 had a positive GCA effect in all and across environments, but was significant only in the Ibadan 1998 environment.

Negative SCA effects were also desirable for resistance. The most resistant cross across environments, TMS30572 x TMS90257, had the

TABLE 3. Mean CMD severity scores and GCA effects for CMD symptom severity of parents in the 3 x 18 NCD II mating scheme, evaluated in three environments

Parent	Across environments		Mokwa 1998		Ibadan 1998		Ibadan 1999	
	Mean	GCA	Mean	GCA	Mean	GCA	Mean	GCA
Female								
TMS30001	1.20	0.13	1.00	0.08	1.15	0.29	1.45	0.02
TMS30555	2.23	-0.04	1.95	-0.02	1.85	-0.11	2.88	0.02
TMS30572	1.55	-0.09	1.10	-0.06	1.94	-0.17*	1.60	-0.04
Mean	1.66		1.35		1.65		1.98	
$LSD_{0.05}$		0.258		0.165		0.165		0.083
Male								
TMS4(2)1425	1.96	-0.06	1.20	-0.04	1.87	0.16	2.80	-0.31*
TMS60142	1.63	-0.20	1.00	-0.19*	1.30	-0.06	2.60	-0.34*
TMS90257	1.48	-0.26*	1.30	-0.14	1.54	-0.01	1.60	-0.63*
TME1	1.48	-0.09	1.10	-0.04	1.58	-0.07	1.75	-0.17
TME2	2.68	0.55*	2.85	0.23*	2.13	0.44*	3.06	0.98*
TME4	1.18	-0.02	1.00	0.02	1.21	-0.08	1.34	-0.01
TME5	1.20	0.06	1.10	0.04	1.20	0.14	1.30	0.01
TME6	1.37	-0.07	1.25	-0.04	1.48	-0.19	1.38	0.01
TME7	1.12	-0.03	1.10	0.05	1.11	-0.05	1.15	-0.07
TME8	1.36	-0.12	1.30	0.00	1.08	-0.06	1.70	-0.29*
TME9	1.15	-0.05	1.05	0.14	1.25	-0.03	1.15	-0.27*
TME10	2.76	0.05	1.93	-0.01	2.78	0.09	3.55	0.07
TME11	1.44	-0.09	1.29	0.12	1.73	-0.22	1.30	-0.18
TME12	1.39	-0.09	1.30	0.03	1.43	-0.12	1.45	-0.20
TME14	1.37	0.06	1.10	0.03	1.30	0.09	1.70	0.06
TME31	2.77	0.20	3.05	-0.03	2.17	-0.04	3.10	0.67*
TME41	3.15	0.01	2.55	-0.10	3.05	-0.11	3.85	0.24*
TME117	3.46	0.17	2.65	-0.05	3.49	0.09	4.25	0.48*
Mean	1.83		1.56		1.76		2.17	
$LSD_{0.05}$		0.238		0.235		0.235		0.144

* Significantly different from zero at the 0.05 probability level
** Significantly different from zero at the 0.01 probability level

lowest mean of 1.48±0.30 and largest negative and significant SCA effect of -0.44±0.09. TMS30555 x TME2, with a mean of 2.57±0.30, had a positive and significant SCA of 0.63±0.09, and was the most susceptible cross across environments. Significant and negative SCA effects were also detected for TMS30001 x TMS60142 in Ibadan 1999 (-0.36±0.09, mean 2.12±0.20) and across environments (-0.28±0.09, mean 1.84±0.30); TMS30001 x TME1 in Ibadan 1999 (-0.40±0.09, mean 2.25±0.20); and TMS30555 x TME12 across environments (0.24±0.09, mean 1.69±0.30).

Significant and positive SCA effects were estimated in all the crosses involving the susceptible male TME2 across environments. Significant and positive SCA effects were also estimated for TMS30001 x TME31, TMS30555 x TME5, TMS30572 x TME117 and TMS30572 x TME31 across environments, and for TMS30001 x TME14, TMS30555 x TME11 and TMS30572 x TME1 in Ibadan 1999.

Negative values for heterosis were also desirable for resistance; however, the average values for mid- and high-parent heterosis were generally positive indicating that the F_1 crosses were generally more susceptible than their mid-parent or high-parents, except in the Mokwa 1998 environment where the F_1 differed from the mid-parent value by -4.95%. The most heterotic cross, which contributed significantly to the average mid-parent heterosis, was TMS30555 x TME41. It had negative and significant mid-parent heterosis of -31.94% across environments and in Mokwa 1998, both mid-parent and high-parent heterosis of -48.44 and -40.51% were significant. The next most heterotic cross was TMS30555 x TME117, which also had negative heterosis in all environments. Its mid-parent heterosis of -50% and high-parent heterosis of -41.03% were significant in Mokwa 1998.

DISCUSSION

A significant correlation between disease incidence, symptom severity and resistance to the cassava mosaic disease is widely known (Fargette *et al.*, 1996). Symptoms of CMD on the field are due to systemic infection from mother plants and whitefly transmission. Although, vector transmission is dependent on whitefly activity, which would vary in different environments, assessment of an accession resistance potential is generally based on its resistance in different environments. Thus, our analyses were based on field resistance across environments (locations and years). The description of the CMD resistance status of the accessions based on the data collected is similar to the CMD classification reported in other studies (Raji, 1995; Lokko *et al.*, 2005). The decision to classify TMS30555 with a score of "2" across, as moderately susceptible was based on a further inspection of the means of the individual environments, where the whole integer of it's mean score in Ibadan 1999 was "3".

Artificial inoculation methods such as bud grafting are used in understanding the mechanisms of resistance (Ogbe *et al.*, 2002). It requires skill and the survival rate ranges to 85%. Recently, a laboratory based artificial inoculation method using a biolistic delivery of a cloned virus into tissue culture plantlets which are then grown under controlled temperature and light conditions, has been used in studies on virus transmission and RNA silencing mechanism of resistance(Ariyo *et al.*, 2003; Vanitharani *et al.*, 2004; Makwarela *et al.*, 2006). Although the biolistic method has been proposed as tools in screening cassava genotypes (Makwarela *et al.*, 2006), in a genetic study such as this with 54 NCD-II crosses with 52 to 934 individuals per cross, each replicated twice in three different environments, the current artificial inoculation methods are not practical. For the grafting method synchronised infection time, skill in inoculating each plant and the less than 100% survival rate are limitations in applying this method to such a study. The high cost in obtaining the biolistic equipment, controlled environment and the risk of loosing genotypes during acclimatisation and transfer to the field, are also major limitations in considering the biolistic method. However, the studies using the artificial inoculation methods give similar classification of resistant and susceptible genotypes as classifications based on field observation across in environment. For instance, Ariyo et al (2003) identified both TME4 and 91/0223 as resistant and Ogbe *et al.* (2002) identified TME117 as susceptible, TMS 30572 as moderately resistance and TMS 30001 as resistant following inoculation,

which are similar to our assessment based on natural infection in different environments.

Our analysis revealed a predominance of additive gene effect based on the significance of the GCA effect of the females and males, the ratios of the mean square components associated with GCA and SCA, and the significant correlation between parental means and GCA. These results are similar to previous reports for resistance to CMD in some cassava accessions (Hahn and Howland, 1972; Lokko *et al.*, 1998).

In this study, a negative and significant GCA effect of a parent indicated a larger contribution towards resistance, while a positive significant value suggested a contribution towards susceptibility. Similarly, a significant and negative SCA of a cross implied that this cross was more resistant than the average GCA of the parents, and significant and positive SCA implied that this cross was more susceptible than the average GCA of the parental accessions.

The overall best general combiner, which contributed the most to resistance, was the resistant improved accession TMS90257 and is recommended to breeding programmes to enhance resistance to CMD. The moderately resistant improved clones TMS30572, TMS60142, and TMS4(2)1425, the resistant landraces TME8 and TME9 with significant and negative GCA, although environmentally dependent, could also be used in breeding for resistance, but the progenies would have to be tested in different environments to select resistant phenotypes. Similarly, the worst general combiner for resistance to CMD was TME2 followed by TME31, TME41, and TME117. In another study conducted in three environments, the landraces TME8, TME9, and TME11 were the best general combiners for resistance to CMD; TMS30001, TMS30555, and TMS30572 were poor general combiners (Lokko *et al.*, 1998). The results of these studies show that the GCA of an accession is dependent on both the environment and the genetic constitution of the population within which it is being tested. Thus, selection of parents in breeding for resistance to CMD cannot be based on the performance of the accession alone. Progeny testing in different environment is essential to facilitate selection of the best parental combinations.

Although two good general combiners were the parents of the most resistant cross TMS30572 x TMS90257, the parents in the other crosses with significant SCA (TMS30001 x TMS60142, TMS30001 x TME1, and TMS30555 x TME12) were not the best general combiners. Furthermore, out of the six crosses that were more susceptible than the average of their parents across environments, only three had one parent as the poor general combiner (TME2). This shows no general relationship between the GCA of a parent and the SCA of its cross. It further emphasises the need for progeny testing to select the best parental combinations for CMD resistance. The significance of individual SCA of crosses, however, also have implications in development new CMD resistant cultivars for target environments, in that progenies could be advanced to yield trial from those crosses which contribute most to resistance in a particular environment.

Based on the significance of the parents' mean squares, which is an indication of the diverse variability, especially among the male parents, it would be expected that a significant amount of heterosis would be obtained. Appreciable average mid-parent heterosis from the parent versus cross-orthogonal contrast was, however, detected in only the individual environments. The significance of the parent versus cross by environment interaction shows that heterosis was environment-dependent. This further illustrates the importance of progeny testing in multiple environments to determine the best parent combinations to enhance resistance to CMD.

ACKNOWLEDGEMENT

This work was supported by the Rockefeller Foundation grant RF 97007 #28 and 2000 FS 137 and managed by the International Institute of Tropical Agriculture (IITA), Ibadan, Nigeria, under its Graduate Research Fellowship programme.

REFERENCES

Akano, A.O., Dixon, A.G.O., Mba, C. Barrera E. and Fregene M. 2002. Genetic mapping of a dominant gene conferring resistance to the

cassava mosaic disease (CMD). *Theoretical and Applied Genetics* 105:521–525.

Ariyo, O. A., Koerbler, M., Dixon, A. G. O., Atiri G. I. and Winter S. 2003. Development of an efficient virus transmission technique to screen cassava genotypes for resistance to cassava mosaic disease. Presented at the Conference on International Agricultural Research for Development, Deutscher Tropentag G_ttingen, October 8-10 2003. http://www.tropentag.de/2003/abstracts/full/268.pdf

Beil, G.M. and Atkins, R.E. 1967. Estimates and specific combining abilities in F_1 hybrids for grain yield and it's components in grain sorghum, *Sorghum vulgare* Pers. *Crop Science* 7:225-228.

Cox, D.J. and Frey, K.L. 1984. Combining ability and selection of parents for interspecific oat matings. *Crop Science* 24:963-967.

Dixon, A.G.O., Bramel-Cox, P.J. and Harvey, T.L. 1990. Diallel analysis of resistance in sorghum to greenbug biotype E: antibiosis and tolerance. *Crop Science* 30:1055-1059.

Fargette, D., Colon, L. T., Bouveau, R. and Fauquet, C. 1996. Components of resistance of cassava to African cassava mosaic virus. *European Journal of Plant Pathology* 102:645 – 654

Fargette, D., Jeger, M., Fauquet, C. and Fishpool L.D.C. 1994. Analysis of temporal disease progress of African cassava mosaic virus. *Phytopathology* 84:91-98.

Gulick, P., Hershey, C. and Esquinas-Alcazar J. 1983. Genetic Resources of cassava and wild relatives. IBPGR report 82/III. International Board for Plant Genetic Resource, Rome, Italy.

Hahn, S.K., John, C., Isoba, G. and Ikoun, T. 1989. Resistance breeding in root and tuber crops at the International Institute for Tropical Agriculture (IITA), Ibadan, Nigeria. *Crop Protection* 8:147-168.

Hahn, S.K. and Howland, A.K. 1972. Breeding for resistance to cassava mosaic disease. In: Terry, E.R (Ed.), pp 37-39. Proceedings, Cassava mosaic workshop, Ibadan, Nigeria. IITA, Ibadan, Nigeria.

Hershey, C. 1987. Cassava germplam resources. In: *Proceedings workshop on cassava breeding: A multidisciplinary review.* The Philippines, 4-7 March 1985. CIAT, Cali Colombia. pp. 1-24.

IITA. 1990. Cassava in tropical Africa. A reference manual. International Institute of Tropical Agriculture, IITA. Chayce Publication Services, United Kingdom 196pp.

IITA. 1997. Annual Report of the International Institute of Tropical Agriculture. IITA, Ibadan, Nigeria.

Jennings, D.L. 1977. Inheritance of linked resistance to African cassava mosaic and bacteria blight. In: Proceedings of the Cassava Protection Workshop, Cali, Colombia, November 1977. CIAT Cali, Colombia. pp. 45-50.

Leuschner, K. 1978. Whiteflies: biology and transmission of African cassava mosaic disease. In: *Proceedings Cassava Protection Workshop, CIAT, Cali, Columbia 7-12 November 1977.* Brekelbaum, T., Belloti, A. and Lozano, T.C. (Eds.), pp. 51-58. CIAT, Columbia.

Lokko, Y., Dixon, A., Offei, S., Danquah, E. and Fregene, M. 2005. Assessment of genetic diversity among African cassava *Manihot esculenta* Crantz accessions resistant to the cassava mosaic virus disease using SSR markers. Genetic Resources & Crop Evolution 00:1–13 (Online 26 Sept. 2005. DOI 10.1007/s10722-005-6841-x).

Lokko, Y., Dixon, A.G.O. and Offei, S.K. 1998. Combining Ability of resistance to the cassava mosaic virus disease. In: Akoroda, M.O. and Ngeve, J.M. (Eds.), pp. 438-442. Root crops in the 21st Century. Proceedings of the 7th Triennial conference of the International Society for Tuber and Root Crops, Africa Branch,. 11-17 Oct. 1998 Cotonou, Benin. IITA, Ibadan, Nigeria.

Makwarela, M., Taylor, N.J., Fauquet, C.M. and Rey, M.E.C. 2006. Biolistic inoculation of cassava (*Manihot esculenta* Crantz) with South African cassava mosaic virus. *African Journal of Biotechnology* 5:154-156,

Nichols, R.F.W. 1947. Breeding cassava for virus resistance. *East African Agriculture and Forestry Journal* 15:154-160.

Ogbe, F. O., Dixon, A. G. O., Atiri, G. I. and Thottappilly, G. 2002. Restriction of Virus

Movement into Axillary Buds is an Important Aspect of Resistance in Cassava to African cassava mosaic virus. *Journal of Phytopathology* 150: 546

Ogbe, F.O., Nnodu, E.C. and Odurukwe, S.O. 1996. Control of African cassava mosaic disease incidence and severity. *Tropical Science* 36:174-181.

Raji, A.A. 1995. Evaluation of pest and disease reactions and trait association in some landraces of cassava. MSc. thesis, University of Ibadan, Ibadan, Nigeria. 86 pp.

SAS Institute. 1999. SAS companion for Microsoft windows environment, version 6, 1st ed. SAS Institute, Cary, North Carolina, USA.

Singh, R.K. and Chaudhary, B.D. 1985. Biometrical methods in quantitative genetics analysis. Kalyan Publications, New Delhi–Ludhiana, India, 3rd Ed. pp. 205-220.

Thresh, J.M., Otim-Nape, G.W., Legg J.P. and Fargette, D. 1997. African cassava mosaic virus disease: The magnitude of the problem. *African Journal of Root and Tuber Crops* 2:13-18.

Vanitharani, R. Chellappan, P., Pita, J.S., and Fauquet, C. M. 2004. Differential Roles of AC2 and AC4 of Cassava Geminiviruses in Mediating Synergism and Suppression of Posttranscriptional Gene Silencing. *Journal of General Virology* 78:9487-9498.

Zhou, X., Liu, Y., Calvert, L., Munoz, C., Otim-Nape, G.W., Robinson, D.J. and Harrison, B.D. 1997. Evidence that DNA-A of a geminivirus associated with severe cassava mosaic disease in Uganda has arisen by inter-specific recombination. *Journal of General Virology* 78:2101-2111.